more, the cell masses do not in every instance completely fill the lymphatics; a central lumen is often preserved which agrees in its appearance, lining, and contents with the lymphatic spaces described. What, then, would appear to have taken place is a growth of cells, either originating from the walls of the spaces or invading the latter from outside? The persistence of the lumen favors the supposition that they come from the walls, and are not the result of an invasion from without.

Additional support for this view of the origin of the cells within the spaces is found in the proliferation actually observed to be going on in such a lymphatic in an uninvaded part of the specimen. From this endothelial-lined wall a growth is forming which at this stage is about as large as a miliary tubercle and which projects into and quite fills, so far as its width extends, the lumen of the vessel. On each side the endothelium is preserved, but goes over gradually into the nodule.

From this description it must be concluded that the tumor has sprung from the endothelium lining enlarged and perhaps new lymphatics in the uterine wall. It is therefore proper to designate it an *endothelioma lymphangiomatodes*.

In conclusion, I wish to express my thanks to Drs. S. Flexner, W. T. Howard, Jr., and Walter R. Lincoln for valuable assistance in the preparation of this paper.

TUBERCULOSIS OF THE AORTA.

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Since the classical researches of Weigert,[1] and his pupil Hanau,[2] on the sources of infection in acute miliary tuberculosis there has been abundant evidence that the walls of the veins may become the seat of tubercle, and the work of Koch[3] and others has shown that the smaller arteries are also quite frequently attacked by the tuberculous process. Instances in which the larger arteries, however, and particularly the aorta, have become implicated in the tuberculous process are so uncommon that two instances which have come under our own observation are here reported.

In reviewing the literature bearing on this subject I have been able to find but few reported instances of the condition, and it would seem worth while to give briefly the main points of each of these. The cases are not all of a similar character, as in three of them the involvement of the aorta was due to direct extension from a tuberculous process originating outside of the vessel; while in the others—and my cases are included in this class—the tubercle was probably due to direct implantation of infective material on the vessel-wall.

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[1] Weigert. Virchow's Archiv, Band lxxvii. and lxxxviii.

[2] Hanau. Virchow's Archiv, Band cviii.

[3] Koch. Mittheilungen aus dem Kaiserl. Gesundheitsamt, Band ii. S. 26.

The cases in which the aortic process was due to a direct extension are those reported by Dittrich,[1] Kamen,[2] and Sigg.[3] Dittrich's case occurred in a boy, aged twelve years, with generalized miliary tuberculosis following chronic glandular involvement; there was present on the posterior wall of the aorta, a short distance above its origin, a bean-sized adherent lymph-gland, corresponding with which there was present on the intima a small oval defect with somewhat elevated edges. Microscopical examination showed that the two processes were continuous, and that the lymph-gland and vessel-wall both showed the histological elements of tubercle, with the presence of many tubercle bacilli.

Kamen's case, which occurred in a cavalryman, aged twenty-four years, was also due to the adherence of a tuberculous gland to the aorta, with subsequent dilatation and rupture of the vessel at the point of adhesion. The process was shown to be tuberculous microscopically, and tubercle bacilli were present in the lymph-gland, though not made out in the wall of the aorta. Ordinary arterio-sclerotic changes were also present in the affected area. Sigg's case occurred in a male, aged sixty-four years, with old apical and intestinal tuberculosis, acute miliary tuberculosis, and arterio-sclerotic changes in the aorta and coronaries. In this case a portion of consolidated lung, containing a cavity filled with caseous material, was adherent to the aorta, which at this point showed bulging of its wall and a perforation of its coats which, externally, was limited by the caseous material filling the cavity, and internally by a thrombus. The microscopical examination showed the usual elements of tubercle, with giant cells and many bacilli, which were present even in the thrombus covering the internal opening in the aorta.

The second group of cases show quite different characteristics, the tuberculous lesions occurring in the form of nodules springing from the intima and projecting into the lumen of the aorta. Weigert[4] reports two cases, presumably of this character, but gives hardly any detail; one of them, he states, was shown to him by Marchand, while the other, which was observed in his laboratory by Huber, is stated to have shown exquisite tubercular structure, with caseous centre and giant cells. Flexner[5] reports the first case of this kind in which a satisfactory description of the lesion is given. It occurred in a male, aged twenty years, who died of general tuberculosis, probably resulting from a tuberculous process in a pulmonary vein. Two and one-half centimetres below the origin of the subclavian artery there was present on the aortic intima a mass which projected into the lumen of the vessel. It was two and one-half millimetres in length and one millimetre in height at its highest point. The mass had a nodular form, and was apparently made up of several single masses; it was pale and translucent. Microscopical examination showed the nodule to be seated directly on the intima and to consist of several masses separated by connective tissue and having caseous centres. Outside of the caseous material the masses were made up of epithelioid cells, a small amount of fibrous tissue, and many leucocytes. A few giant cells were present, and tubercle bacilli were also demonstrated in small numbers. There were no arterio-sclerotic changes in the aorta in this case. The case of Hanot and Lévy[1] occurred in a man, aged sixty-one years, who died of acute miliary tuberculosis. There was present in the upper part of the thoracic aorta a tubercular nodule; it was single, round in shape, grayish, of firm consistency, and the size of a pinhead. It projected slightly into the lumen of the vessel. The microscopic examination showed a caseous centre and about this tuberculous structure containing a few giant cells. Tubercle bacilli were present in the nodule, but only in small numbers, judging by the illustration which accompanies the article. The middle and external coats were free from evidences of tubercle, and no arterio-sclerotic changes were present. There was a small inflammatory area at one point in the adventitia.

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[1] Dittrich. Prager Zeitschrift für Heilkunde, 1898, S. 97.

[2] Kamen. Ziegler's Beiträge, 1895, S. 416.

[3] Sigg. Beiträge zur Lehre der Acuten Miliartuberculose. Inaug. Discuss. Zürich, 1896.

[4] Weigert. Virchow's Archiv, Band lxxxviii. S. 360.

[5] Flexner. Johns Hopkins Hospital Bulletin, 1891, p. 120.

Stroebe's[2] case is reported as one of tuberculous aortitis, but from his description it would not seem to differ materially from those already cited. The deeper parts of the intima seem, perhaps, to have been more involved than in Flexner's or Hanot and Lévy's cases. In this case there was present in the ascending aorta, two centimetres above the sinus of Valsalva, a small excrescence seven millimetres in height and three to four millimetres in thickness. It was irregular and nodulated and had a pedunculated character, the outer enlarged portion being covered by a partly organized and partly fresh clot. Histological examination showed the nodule to be made up peripherally of epithelioid cells and polynuclear leucocytes; centrally, of caseous material; the apex was capped by a partly organized clot. Numerous tubercle bacilli were present in the nodule and extended to its periphery in one place so as to be in actual contact with the blood-stream. The media and intima were unaffected except that in the neighborhood of the tuberculous process a portion of the media was necrotic, and there were collections of polynuclear leucocytes in the neighborhood of the vasa vasorum. No arterio-sclerotic changes were present in the vessel wall. Stroebe, in his paper, includes among probable cases of the affection that of Zahn,[1] which was described as a case of aortitis verrucosa; Marchand also believes this to be a case of aortic tuberculosis. Zahn himself, however, who was present at the demonstration of Stroebe's specimens at the Brunswick Naturalists' Society, states flatly that the case was not one of tuberculosis, and hence we will not include any description of it.

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[1] Hanot and Lévy. Archives de Médecine Expérimentale, 1896, p. 784.

[2] Stroebe. Cent. für allg. Path. und Path. Anat., 1897, S. 998.

Of my own two cases, the first was observed in the Pathological Laboratory of the Johns Hopkins University and Hospital, and I am indebted to Prof. W. H. Welch for permission to report it; for the second case I am indebted to Dr. George E. Gorham, of Albany, N. Y., in whose practice it occurred.

CASE I.—Colored man, aged forty-five years. The anatomical diagnosis was as follows: Chronic tuberculosis of the peritoneum; acute miliary tuberculosis of the lungs, liver, spleen, kidneys, intestine, adrenals, pancreas, trachea, bronchi, epididymis, testicle, bladder, brain membranes, bone-marrow, and aorta; cloudy swelling of the liver and kidneys; arterio-sclerosis; slight coronary artery disease; slight chronic interstitial myocarditis.

The following are the notes on the aorta:

Macroscopical. The aorta shows a patchy atheroma throughout in a moderately advanced fatty stage. In the descending aorta three nodules are to be seen, all of them apparently below the diaphragm; two of them appear as pinhead-sized, gray, translucent nodules projecting above the surface of the intima and sharply localized; the third one has a yellowish tinge, is slightly larger, and is apparently situated in the vessel-wall.

Microscopical. Each nodule appears as a small, rounded elevation seated on the intima of the aorta. The endothelium covering the intima can be traced as far as the projecting nodule, but does not extend over this. The intima does not show any thickening where the nodule occurs, but in other places in the section it is distinctly thickened and, in its deeper portions is at times devoid of nuclei, and evidently necrotic. The nodule affects only the most superficial layers of the intima; in fact, it has almost the appearance of being stuck on to its inner surface. The parallel arrangement of the intimal nuclei can be traced even in its most superficial parts under almost the entire area involved by the nodule; only at one end is there slight cellular infiltration affecting the superficial intimal structure. The media shows in places slight changes in the form of an infiltration of round cells between the muscle cells in the neighborhood of the vasa vasorum; these changes are most marked in the portions of the media lying beneath the thickened areas in the intima.

The nodule mentioned above is made up almost entirely of cells of the epithelioid type, with long or irregular-shaped nuclei. The central portion is almost free from nuclei and has the usual appearance of caseous material. An occasional cell, with a round, darkly-staining nucleus is seen in the portion of the nodule next to the intima, but no such cells are to be made out in the portion next the blood-stream. No definite giant cells are seen, though in one or two places there are clumps of epithelioid nuclei suggesting their formation. No remains of blood-pigment are seen in the nodule. Sections stained with carbol-fuchsin and decolorized with acid alcohol showed a few tubercle bacilli. The lymph glands in the immediate neighborhood contain definite caseous tubercle nodules, with giant cells, but are nowhere adherent to the aorta.

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[1] Zahn. Virchow's Archiv, Band lxxii.

CASE II.—White woman, aged fifty years. The anatomical diagnosis was as follows: Old caseous tuberculous focus at the apex of the right lung, with softening, miliary tuberculosis of both lungs and the peritoneal covering of the spleen; miliary tubercles of the spleen and liver; acute swelling of the spleen; caseous tuberculosis of the kidneys; fresh tuberculous ulcers in the stomach and ileum; tuberculosis of the descending aorta.

The following is the abstract from the protocol referring to the aorta:

Macroscopical. At one point in the descending aorta there is present a small, pinhead-sized excrescence. It is rather firm in consistency, gray and translucent in appearance and with a finely-lobulated surface, being apparently formed by the growing together of three or four small circular nodules. It springs from the intima by a moderately broad base, but is slightly constricted at a short distance from its origin, which gives it a polypoid appearance. The aorta shows a number of patches of fatty and calcareous atheroma.

Microscopical. The excrescence described in the naked-eye description is seen as an irregular elevation on the intima. The elevation springs from a broad base and varies in shape in different sections. In some it is rounded, but in others shows definite excrescences corresponding to those seen with the naked eye. It is made up of a cellular and a necrotic portion. The cellular portion is situated at the periphery of the nodule, and is made up almost exclusively of irregular cells of an epithelioid type. In places in this portion an occasional small round cell is seen, and in one section a typical giant cell is present. The entire periphery of the nodule is not equally cellular, for while there are many cells in the portions of the periphery abutting on the blood-stream, the portion lying against the intima shows only a very few epithelioid cells lying in the innermost portions of this coat; in fact, the intima seems scarcely at all invaded, and the nodule in this, as in the previously described case, has almost the appearance of having been stuck on to the intima. The central portion is almost free from nuclei, contains a few nuclear fragments, and has the usual appearances of caseous material. The epithelial layer of the intima apparently stops short at the nodule, and does not cover it. The intima is apparently not thickened beneath the nodule, nor does it show any thickening elsewhere in the specimen. The media and adventitia show no changes. Sections stained with carbol-fuchsin and methyl-blue show innumerable tubercle bacilli in the cellular portions of the nodule (a few in the caseous portion). They have no definite arrangement. Nothing resembling altered blood-pigment is to be seen in the nodule.

Aside from the extreme rarity of the lesion, the last group of cases are of interest with regard to the method of origin of the tubercles. It may be assumed that tubercles of the aorta can arise either by direct transplantation of infective material from the blood-current or from the entrance into the vasa vasorum of tubercle bacilli. The probability of the latter mode of origin is almost certainly negatived in all of the reported cases belonging to this class by the situation of the lesion; in all but one of the cited instances the media did not show any changes that could be regarded as tuberculous in nature, and in Stroebe's case there was only a slight amount of necrosis of the innermost portion of this coat. According to the observations of Plotnikow,[1] the vasa vasorum of the aorta extend only half-way into the media from its external surface, so that the possibility of infection in this way in these cases is very slight. An origin from infective material carried in the blood-stream seems much more probable in all these cases; in our two cases the most striking thing in the sections was the extremely slight involvement of even the most superficial layers of the intima, the tubercles looking almost as if they had been stuck on to this coat, so slightly was it affected; the illustration in Hanot and Lévy's case would lead one to suppose that here, also, only the superficial portions of the intima were affected, and the description in Flexner's case gives one the same impression. The method of infection here is probably similar to that which occurs in some forms of endocarditis and aortitis. The experiments of Ribbert,[2] of which Stroebe speaks, show that, in the case of the endocardium, infection of the valves can take place without any previous disease or traumatic influence, though in these experiments foreign bodies in the shape of fine particles of potato were introduced into the circulation with the bacilli. In the experiments of Boinet and Romary,[3] these authors were at times able to set up changes in the aorta resembling arterio-sclerosis by the mere injection of bacteria, without accompanying trauma or the presence of foreign particles. It is presumable that the pre-existence of some lesion of the intima would favor the development of the tubercles, though there was no apparent indication that such changes were present in any of the cases under discussion. The fact that the existence of a previous lesion may favor the development of tuberculosis is shown by the case of Cordua,[4] where an œsophageal carcinoma became infected with tuberculosis, and the case of Breus,[5] where both gastric and œsophageal cicatrices became similarly infected. The occurrence of arterio-sclerosis in connection with the tuberculous lesion in some of the cases is certainly suggestive in this connection; but in several of the cases the individuals affected were very young and showed absolutely no change in the vessel-walls, while in other cases, as in our own, though arterio-sclerosis was present in the vessel-wall in places, none was to be observed in the immediate neighborhood of the tubercle nodules. It is conceivable that in cases of this sort, in which large quantities of bacterial toxins are circulating in the blood-stream, these might be able to cause localized areas of lessened resistance in the vessel-wall and prepare it for the lodgement of the tubercle bacillus. That bacterial toxins, without the presence of the bacteria themselves and without accompanying traumatism, are well able to cause changes in the aorta is shown by the researches of Boinet and Romary,[1] who were able to produce definite lesions by the injection of the toxins of tetanus and of diphtheria. Direct infection from the lymph vessels is hardly to be thought of, the current being in the wrong direction to convey infection from the neighboring glands, and the vessels in all probability reaching only the same distance into the vessel-wall as do the bloodvessels—*i. e.*, to the middle of the media.

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[1] Plotnikow, cited by Stroebe. [2] Ribbert. Fortschritte der Medicin, 1886, S. 1.

[3] Boinet and Romary. Archives de Médecine Expérimentale, 1897, p. 902.

[4] Cordua. Arbeiten a. d. patholog. Institut in Göttingen, 1893.

[5] Breus. Wiener med. Wochenschrift, 1878, No. 11.

The possibility of these tubercle nodules being due to the invasion of small parietal thrombi by the tubercle bacillus must be thought of, as invasion of thrombi by tubercle bacilli has been observed in connection with heart lesions in some instances; but in none of the reported cases was there evidence to support this theory. In Stroebe's case there is distinct mention made of the fact that nothing like changed blood-pigment was present in the tubercle nodules, nor could we find any evidence of such in our cases. The presence of a small amount of fibrin in the tubercles, such as occurred in Stroebe's cases, is no evidence of thrombotic origin, for Falk[2] has shown that a fair percentage of tubercles contain fibrin at some stage in their development. The results of the experiments of Boinet and Romary on the production of aortic inflammatory lesions lead us to hope that in the future the origin of aortic tubercles may be made clearer by similar methods.

COMBINED TYPHOID AND MALARIAL INFECTION.

REPORT OF A CASE AND REVIEW OF THE LITERATURE, WITH SPECIAL REFERENCE TO THE SO-CALLED TYPHO-MALARIAL FEVER.

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For the past half-century there has been much controversy among medical men on the question of the coexistence of typhoid and malarial

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[1] Boinet and Romary. Archives de Médecine Expérimentale, 1897, p. 902.

[2] Falk. Virchow's Archiv, Band cxxxix. S. 339.